The claimed invention is:

1. A method of inducing IBD-like symptoms in a mammal, wherein the mammal (i) is a non-human mammal not expressing a functional mdr1a gene product, and (ii) is subjected to elevated chlorine concentrations.

2. A method of inducing IBD-like symptoms in a mammal, wherein the mammal is treated with an inhibitor of the mdr1a gene product and is subjected to elevated chlorine concentrations.

3. The method of claim 1 or claim 2, wherein the elevated chlorine concentrations are administered by supply of chlorinated drinking water to the mammal.

4. The method of claim 3, wherein the concentration of chlorine in the drinking water is above about 1ppm.

5. The method of claim 1, wherein the mammal is a transgenic mdr1a$^{-/-}$ knockout mouse.

6. The method of claims 1 or 2, wherein IBD-like symptoms occur in about 100% of the mammals.

7. A method of screening a candidate compound for its efficacy in ameliorating the symptoms of IBD, the method comprising the following steps:
   (a) administering the candidate compound in a vehicle to a first non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited, subjected to elevated chlorine concentrations;
   (b) administering the vehicle without the candidate compound to a second non-human mammal not expressing a functional mdr1a gene product or wherein the

mdr1a gene product or its expression is effectively inhibited, subjected to elevated chlorine concentrations; and

(c) comparing the symptoms of IBD in the mammal(s) of steps (a) and (b),

wherein a decrease in symptoms of IBD in the mammal(s) of step (a) as compared to the mammal(s) of step (b) indicates efficacy of the compound.

8. A method of screening a candidate compound for its efficacy in preventing or delaying the development of IBD, the method comprising the following steps:

(a) administering the candidate compound in a vehicle to a first non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited, before the onset of the disease in said mammal;

(b) administering the vehicle without the candidate compound to a second non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited, before the onset of the disease in said mammal;

(c) subjecting the mammal(s) of step (a) and step (b) to elevated chlorine concentrations; and

(d) comparing the onset of any symptoms of IBD in the animals,

wherein a delay in or prevention of the onset of symptoms of IBD in the mammal(s) treated with the candidate compound in a vehicle compared to the mammal(s) treated with the vehicle without the candidate compound indicates efficacy of the compound.

9. A method of screening for genes that may be involved in the pathogenesis of IBD comprising the following steps:

(a) subjecting a first non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited to elevated chlorine concentrations;

(b) not subjecting a second non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited to elevated chlorine concentrations;

(c) making RNA preparations from the intestine from both the mammals of step (a) and of step (b) after the desired time interval; and

(d) comparing the RNA samples,

wherein a RNA which shows a difference in these samples indicates a gene that may be implicated in the pathogenesis of IBD.

10. A method of screening for genes that may be involved in the pathogenesis of IBD comprising the following steps:

(a) subjecting a first non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited to elevated chlorine concentrations;

(b) subjecting a second non-human mammal expressing a functional mdr1a gene product to elevated chlorine concentrations;

(c) not subjecting a third non-human mammal not expressing a functional mdr1a gene product or wherein the mdr1a gene product or its expression is effectively inhibited to elevated chlorine concentrations;

(d) not subjecting a fourth non-human mammal expressing a functional mdr1a gene product to elevated chlorine concentrations;

(e) making RNA preparations from the intestine from each of the mammals of steps (a) to (d) after the desired time interval; and

(f) comparing the RNA samples,

wherein a RNA which shows a difference between the samples of step (a) and step (c), but not a similar difference between the samples of step (b) and step (d), is a gene that may be implicated in the pathogenesis of IBD.

11. The method of claim 9 or 10, wherein the RNA samples are compared by expression profiling.

12. The method of claim 11, wherein the expression profiling is performed by microarray analysis.

13. The method of one of claims 9 or 10, further comprising the step of identifying the human homologue of the identified gene.

14. The method of one of claims 7 to 10, wherein the elevated chlorine concentrations are administered by supply of chlorinated drinking water to the mammal and wherein the concentration of chlorine in the drinking water is above about 1 ppm.

15. The method of claims 9 or 10, wherein the step of not subjecting the mammal to elevated chlorine concentrations is carried out by supplying only unchlorinated drinking water to the mammal and not otherwise supplying an elevated chlorine concentration to the mammal.